BC Environment

A REVIEW OF THE ENVIRONMENTAL IMPACT AND TOXIC EFFECTS OF DDAC

N.D. Henderson
Prepared for:
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SUMMARY

This paper summarizes the physical, chemical, environmental, and toxicological properties of didecyldimethylammonium chloride (DDAC), an active ingredient in several antisapstain products used by the Canadian lumber industry.

Laboratory studies have shown that DDAC is photolytically and hydrolytically stable, and immobile in soil. These studies also show that DDAC is resistant to microbial degradation, although earlier study results showed complete degradation within 48 hours when DDAC was incorporated into mixed bacterial soil and sewage cultures. Differences in studies and findings suggest that the composition of the microbial population is an important variable in the degradation of DDAC. The concentration of DDAC also appears to be an important variable since in another laboratory study, high primary degradation was found at a DDAC level of 5 ppm while poor ultimate degradation was observed at 15 ppm. Predicted half-lives in the soil and aquatic environments are 3 years, and 17 to 23 years, respectively. These half-lives are calculated from studies of much shorter duration (i.e. 30 days, one year) than the calculated half-lives.

In two leaching studies using DDAC treated lumber, DDAC concentrations between 48 and 73.2 ppm were found in the initial leachates. The concentrations of DDAC in later leachates in both studies were in the 6 ppm range.

In rats, DDAC is minimally absorbed from the gastrointestinal tract. It is excreted in the urine and feces, and a negligible amount is retained in the body after one week. It is moderately to very toxic to mammals orally and dermally, depending on formulation and according to toxicity scales used in this report. It is a severe eye and skin irritant.

Using toxicity scales derived for this paper and others, DDAC is slightly to very toxic to salmonid species, depending on formulation. DDAC is also extremely toxic to mysid shrimp, and is highly phytotoxic to an alga and a duckweed. In a bluegill bioconcentration study, DDAC was found to reach steady-state levels rapidly in fish tissue and to depurate rapidly (7 to 14 day half-life) following re-exposure to untreated water. The bioconcentration factor in edible fish tissue was determined to be 38x, suggesting little likelihood for bioaccumulation in fish tissue.

DDAC has displayed no carcinogenic, mutagenic, or teratogenic activity in laboratory tests. The observed severe irritation effects observed in laboratory animals suggests that acute human exposure may result in severe effects through skin and eye contact, or through ingestion. The use of DDAC in the workplace should not pose a significant health risk provided proper protective equipment is worn and proper safety practices are observed.

TABLE OF CONTENTS

		PAGE
Summary		2
Table of Contents		3-5
1.0	INTRODUCTION	6
2.0	HISTORICAL AND BACKGROUND USES	6-7
3.0	PHYSICAL AND CHEMICAL PROPERTIES	7-8
4.0	ENVIRONMENTAL IMPACT	8-11
	4.1 Environmental Sources	8
	4.2 Environmental Distribution	8
	4.3 Environmental Behaviour	8-11
	4.3.1 Photolysis	9
	4.3.2 Hydrolysis	9
	4.3.3 Soil/Sediment Adsorption/Desorption	9
	4.3.4 Biotransformation and Soil Metabolism	9-10
	4.3.5 Aerobic Aquatic Metabolism	10
	4.3.6 Anaerobic Aquatic Metabolism	10
	4.3.7 Leaching from Wood	10-11
5.0	PHARMACOKINETICS	11-13
	5.1 Absorption, Transport, and Distribution	12
	5.2 Metabolism and Excretion	12-13
	5.3 Placental and Milk Transfer	13
	5.4 Pharmacokinetics of DDAI	13

		PAGE
6.0	TOXICOLOGICAL EFFECTS	13-20
	6.1 Human Toxicity	13
	6.2 Mammalian Toxicity	13-17
	6.3 Aquatic Toxicity	17-18
	6.3.1 Bioconcentration	18-19
	6.4 Avian Toxicity	19
	6.5 Phytotoxicity	19-20
	6.6 Comparative Toxicity	20
7.0	CARCINOGENICITY	20
8.0	MUTAGENICITY	20-21
9.0	TERATOGENICITY AND REPRODUCTIVE EFFECTS	21-23
10.0	IMPACTS OF OCCUPATIONAL EXPOSURE	23-24
11.0	FOOD USE	24
12.0	ANALYTICAL METHODS OF DETECTION	24-25
13.0	EXISTING LEGISLATION	25
14.0	CONCLUSION	25-26
15.0	REFERENCES	27-36
Table 1:	A comparison of toxicity values of antisapstain chemicals	37
APPENDIX A -	Databases searched for information on DDAC	38
APPENDIX B -	Products containing DDAC, and their manufacturers	39

PAGE

APPENDIX C - Mammalian lethal toxicity classes for chemical compounds 40

APPENDIX D - Aquatic lethal toxicity classes for chemical compounds 41

APPENDIX E - Calculation of an Acceptable Daily Intake (ADI) of DDAC for Humans 42

APPENDIX F - Derivation of stormwater effluent criteria for DDAC 43

APPENDIX G - Calculation of an air emission guideline for DDAC 44-45

1.0 INTRODUCTION

Large volumes of chlorophenol compounds have previously been used in Canada to preserve and protect wood from attack by various pests. Concerns about acute aquatic toxicity, occupational impacts, and presence of hazardous impurities, including dioxins and furans, in chlorophenols are among the reasons for dissatisfaction with their use (5). The Canadian lumber industry has thus phased out the use of chlorinated phenols for sapstain control, and the use of alternative chemicals has increased.

Currently there are several chemicals registered with Agriculture Canada which may be used for the protection of freshly cut lumber, including 3-iodo-2-propynyl butyl carbamate (IPBC), didecyldimethylammonium chloride (DDAC), sodium carbonate, sodium borate, azaconazole, and copper-8-quinolinolate (Cu-8).

The purpose of this paper is to review and evaluate available information concerning the physical and chemical properties, environmental behaviour, and toxicological effects of DDAC. This chemical is an active ingredient in a number of antisapstain formulations, including NP-1, Timbercote II, F-2, and Ecobrite III. Information on NP-1 is included in a separate report (39), while information on the other formulations is included where available. Information on non-formulated DDAC is also included. Companies marketing products containing the chemical were contacted for information, and a literature search of selected computerized databases was carried out (Appendix A).

2.0 HISTORICAL BACKGROUND AND USES

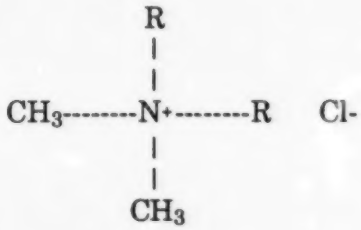
DDAC is a quaternary ammonium compound (QAC), and is a member of a group of such chemicals well known for their germicidal, fungicidal, and algicidal activity. QACs were first synthesized in the late 1800's, and their bactericidal properties were reported about two decades later. By 1935, their germicidal and antiseptic potential had been characterized. Since that time, extensive research on toxicity and side effects has been carried out. Presently, formulations containing between 0.01 and 1.0% QACs are used as antiseptics, bactericides, fungicides, sanitizers, and deodorants (21). QACs are also popular disinfectants for utensils, containers, and other instruments used in restaurants, dairies, food plants, laundries, and operating rooms (21).

DDAC formulations have long been used as antimicrobials and antiseptics in hospital and industrial settings (1). Bardac 2280, 80% DDAC in an alcohol/water base, is one such product. Manufactured by Lonza Inc., it has found

found wide use as a disinfectant, sanitizer, household cleaner, mildew preventative in commercial laundries, and microbicide in water treatment (7). DDAC is currently registered in Canada as an anti-microbial, and as an active ingredient in the antisapstain products NP-1, F-2, Timbercote II, and Ecobrite III (66). Appendix B lists registered products containing DDAC, and their manufacturers.

3.0 PHYSICAL AND CHEMICAL PROPERTIES

The chemical structure of DDAC is shown below.



($R = C_{10}$)

(CAS No. 7173-51-5)

DDAC is a nonvolatile, photolytically stable salt which is highly soluble in water (2). Its octanol/water partition coefficient is reportedly zero (2, 64), so bioconcentration and bioaccumulation are not expected to occur. Data on other physical properties such as boiling point, freezing point, and density were not located. Bardac 2250, manufactured by Lonza Inc., contains 50% DDAC, 9-11% ethanol, 32.8-40% water, and 2-5% other minor constituents. It has a flash point of 42°C, a specific gravity of 0.927 at 25°C, and a pH of 6.5 to 9.0. It is soluble in water, ethanol, and glycols, and is dispersible in hydrocarbons. Bardac 2280 contains 80% DDAC, 9-11% ethanol, 1.8-8.55% water, and the remainder other minor constituents (70). Its flashpoint is 43°C, and its specific gravity is 0.891 at 25°C. The pH and solubility characteristics are similar to Bardac 2250 (79).

Timbercote II is composed of 20% DDAC, 2.5% ethyl alcohol, and a synthetic resin in aqueous emulsion (40). It has a boiling point of 100°C, a freezing point of 0°C (30), a pH of 5 to 7, and a specific gravity of 0.989 (40). It has poor freeze/thaw stability and its vapour pressure is unknown (69). The resin component is a vinyl acetate copolymer emulsion with a pH of 4 to 6 and a specific gravity of 1.05-1.15. It is a non-flammable white fluid with a mild acetic acid

odour. Hazardous decomposition products of this copolymer emulsion include carbon monoxide, carbon dioxide, and possibly traces of vinyl acetate (30).

F-2, composed of 11.4% DDAC and 16.8% borax, is a translucent amber liquid emulsion with a mild detergent odour. It has a pH between 7 and 8, a specific gravity of 1.077, and a flashpoint greater than 90°C. It is stable at 25 C, and has some freeze-thaw stability (64). F-2 is 100% soluble in water at 20°C, and is insoluble in oil.

Because NP-1 contains another active ingredient in addition to DDAC, the physical and chemical characteristics of NP-1 are included in another review (39). Information on Ecobrite III was not available.

One source states that incineration of DDAC is regarded as safe (22), although temperature and other details of incineration were not stated. Another source states that potentially toxic fumes may form and include carbon dioxide, carbon monoxide, ammonia, nitrogen oxides, and hydrogen chloride gas (2). Dioxins reportedly do not form (49). Further details on incineration of DDAC were not available.

4.0 ENVIRONMENTAL IMPACT

4.1 Environmental Sources

DDAC is not known to be naturally occurring, so all DDAC in the environment is expected to be from human sources. Such sources could include spills and other unpermitted discharges, permitted discharges from commercial facilities using the chemical, and discharges from products treated with DDAC, including wood, laundered items, and sterilized equipment and utensils.

4.2 Environmental Distribution

No studies on levels of DDAC in ambient air, water, or soil were located.

4.3 Environmental Behaviour

Several laboratory-based environmental fate studies, including aerobic soil metabolism, and aerobic and anaerobic aquatic metabolism studies, have been completed and submitted by the manufacturer. These studies are summarized below. Field studies on the environmental fate of DDAC are also being planned by the manufacturer, such that the behaviour of the compound in real environments (as opposed to laboratory environments) may be better understood. These studies should be completed in 1992 (99).

4.3.1 Photolysis

A study conducted with carbon-14 labelled DDAC at 25°C in aqueous buffered solution (pH 7) found the chemical to be stable to photolysis (12). A xenon light source, simulating natural sunlight, continuously illuminated the solution for 30 days. At the end of this time, no significant degradation of the test compound had occurred. In the presence of the photosensitizer acetone, approximately 7% of the parent compound was degraded. The photolysis rate constant and half life for this system were calculated to be 0.00304/day and 227 days, respectively. An accurate measure of the photolytic half life could not be determined since no significant degradation occurred.

4.3.2 Hydrolysis

A 30 day hydrolysis study using carbon-14 labelled DDAC at 25°C in aqueous solutions buffered to pH 5, 7, and 9 found the chemical to be stable within this pH range (13). All experiments were conducted at a nominal test concentration of 10 ug/ml, under sterile conditions and in darkness. An accurate measure of the hydrolytic half life could not be determined since no significant degradation occurred.

4.3.3 Soil/Sediment Adsorption/Desorption

A soil adsorption/desorption study conducted at 25°C in the dark with four soil types found that the chemical was immobile in all soils tested (14). The study was conducted at a 1:200 soil to water ratio using four nominal concentrations of 0.70, 3.50, 5.25, and 7.00 ppm DDAC in sand, sandy loam, silty clay loam, and silt loam (0.25, 0.90, 2.05, and 2.1% organic carbon, respectively). The study concluded that DDAC is essentially immobile in soil. Consequently, DDAC in soil would be unlikely to contaminate groundwater.

4.3.4 Biotransformation and Soil Metabolism

Biotransformation is expected to be the main route of dissipation of DDAC in the environment (2). An older, completed study indicated that rapid and complete degradation of DDAC occurs within 48 hours when low concentrations (10 ppm) are exposed to mixed bacterial cultures obtained from soil and sewage (34). A laboratory examination of the biodegradability of DDAC found a high primary degradation at a level of 5 ppm, but a poor ultimate degradation at 15 ppm (65).

In contrast to the above studies, a one-year aerobic soil metabolism study recently submitted by the manufacturer found that DDAC is stable to microbial

degradation (93). The study used a microbially active sandy loam treated at a nominal concentration of 10 ppm of ^{14}C-DDAC, and incubated in the dark at 25°C. Soil samples collected at 0, 1, 3, 7, 14, 31, 61, 92, 123, 182, 273, and 365 days after dosing were extracted and analyzed for ^{14}C-DDAC. Following the incubation period, 72.9% of the dosed radioactivity remained as parent compound. An aerobic half-life of 1,048 days (~3 years) was calculated, based on first-order degradation. However, an accurate measure of the soil half life could not be determined since no significant degradation occurred. Accumulative volatiles amounted to 1.95% of the dosed radioactivity at the end of the study.

4.3.5 Aerobic Aquatic Metabolism

Data from this study also indicate that ^{14}C-DDAC is stable to microbial degradation (94). Microbially active pond water and sediment treated to a nominal concentration of 10 ppm ^{14}C-DDAC were incubated for one year under dark, aerobic conditions at 25°C. Flooded sediment samples collected at 0, 1, 3, 7, 14, 31, 61, 92, 123, 182, 273, and 365 days after dosing were extracted and analyzed for ^{14}C-DDAC. The water layers from the flooded sediment samples were also analyzed for ^{14}C-DDAC.

Following the incubation period, a mean of 88.5% of the dosed radioactivity remained as parent compound. A half-life of 8,365 days (~23 years) was calculated, based on first-order degradation. Again, an accurate half-life could not be determined because of the low level of degradation. At the end of the study, accumulative volatiles amounted to 4.51% of the dosed radioactivity.

4.3.6 Anaerobic Aquatic Metabolism

This study again found ^{14}C-DDAC to be stable to microbial degradation (95). Microbially active water and sediment treated to a nominal concentration of 10 ppm ^{14}C-DDAC were incubated under dark, anaerobic conditions at 25°C for one year. Flooded sediment samples collected at 0, 1, 3, 7, 14, 31, 61, 92, 123, 182, 273, and 365 days after dosing were analyzed for ^{14}C-DDAC. The water layer from the flooded sediment samples was also analyzed.

Following the incubation period, a mean of 90.8% of the dosed radioactivity remained as parent compound. A half-life of 6,218 days (~17 years) was calculated, based on first-order degradation. Again, this half-life is an estimate and not an accurate measure since no significant degradation occurred during the study.

4.3.7 Leaching from Wood

Leaching of DDAC from treated wood has been tested using F-2, Timbercote II, and NP-1. In the first F-2 study (20), significant quantities of borate and DDAC

(780 mg/m^2 and 112 mg/m^2, respectively) were lost from the surface of treated wood under conditions of simulated rainfall. Unfortunately, potential levels of the chemical in stormwater and the detection limits for this experiment were not reported.

In the second F-2 study (61), unseasoned hem-fir lumber was spray-treated with a 14% active ingredients F-2 solution (~6% DDAC). A rotary lawn sprinkler provided a constant and uniform rainfall of about 5.5 mm/hr to each of three packages. Leachates were collected at the tilted end of each package from 30 minutes after the onset of sprinkling, to completion of the study after eight days.

Concentrations of DDAC measured in the leachates fell rapidly from an initial level of 48 ppm to below 10 ppm after 12 hours, and to about 6 ppm after 24 hours. Keeping the package protected for 24 hours before simulated rainfall greatly reduced the initial rates of leaching to about 15 ppm, but final leaching rates after 48 hours were similar to those from the unprotected package. After 48 hours of rainfall (264 mm), about 6.4% of the DDAC originally applied had been leached from the lumber surfaces of both the unprotected package and the package stored under cover for 24 hours before leaching.

The Timbercote II study (62) measured DDAC concentrations in leachate from two treated and two untreated packages of lumber after seven rainfall events over 18 days. Artificial rainfall was not used for this study to avoid the possibility of chlorine contamination from tap water in the leachate samples. Results indicate that DDAC leached from Timbercote II treated lumber: DDAC concentrations of 73.2 and 65.8 ppm were recorded during the first two leaching cycles. After the seventh leaching cycle, a DDAC concentration of 6 ppm was measured in the leachate.

An NP-1 leaching study done by government and industry has also been completed (92). However, a final report was not available to the Ministry at the time of finalization of this document, so results cannot be summarized here.

5.0 PHARMACOKINETICS

One preliminary and three large-scale pharmacokinetic experiments on rats have been performed. These studies determined absorption, distribution, metabolism, and excretion patterns following the administration of carbon-14 labelled DDAC (23). The preliminary experiment collected data on the amount of ^{14}C eliminated in the expired air following acute oral administration of ^{14}C-DDAC. Results of this experiment and results of administering DDAC as a single low oral dose, as a repeated low oral dose, or as a single high oral dose are summarized below.

In the preliminary experiment, four rats (2 male, 2 female) were fasted for 18 hours, then administered 5 mg/kg ^{14}C-DDAC. Animals were immediately placed in individual glass metabolism cages, and expired CO_2 was collected and analyzed for radioactivity.

Eighteen hour fasted rats (5 male, 5 female) were used in each of the large-scale experiments. Animals were dosed with either 5 mg ^{14}C-DDAC/kg (low dose groups), or with 50 mg/kg (high dose group). Rats in the repeated low oral dose experiment were fed diets containing 34 ppm DDAC for 14 days prior to administration of a single dose of 5 mg/kg ^{14}C-DDAC. For all groups, urine and feces were collected at predetermined intervals and subsequently analysed for ^{14}C content. Seven days after dose administration, rats were euthanized and selected tissues and organs were analyzed for radioactivity.

5.1 Absorption, Transport, and Distribution of DDAC

Because DDAC is highly ionic, it is not expected to be well absorbed from the gastrointestinal tract after oral administration. Excretion results (see below) are consistent with this expectation. Transport characteristics of the chemical were not documented, but tissue and carcass analysis revealed that only a negligible amount (0.003-0.675%) of dosed radioactivity remained in the body after 7 days.

5.2 Metabolism and Excretion of DDAC

The preliminary study (23) showed that little or no radioactivity (0.041-0.054% of dose) was excreted as $^{14}CO_2$ during the 24 hours following oral administration of ^{14}C-DDAC. This indicates that the ^{14}C radiolabel was in a stable portion of the DDAC molecule.

In all of the large-scale experiments, 89-99% of the recovered radioactivity was found in the feces, and less than 2.5% was found in the urine. This finding is consistent with the predicted low absorption of the chemical after oral administration. The pattern of excretion of radioactivity in male and female rats in all dosage groups was similar.

A further study to determine the metabolic profile of ^{14}C residues in fecal samples involved dosing 5 male and 5 female rats with 50 mg ^{14}C-DDAC/kg (24). Following extraction and clean-up procedures, the metabolic profile of ^{14}C-residues in the feces was determined and compared with that of animals in the large-scale studies (23). The pattern of excretion was again similar between the sexes, although a dose-dependent metabolism was observed in females: more parent compound was metabolized in the single low oral dose than in the single high oral dose.

The metabolic process for DDAC in the rat was found to involve oxidation of the decyl sidechain to a variety of oxidative products. Evidence seems to favour initial hydroxylation of the carbon next to the terminal carbon, followed by formation of a hydroxyketone. The four major metabolites found in this study were more polar and presumed to be less toxic than the parent compound, although their chemical structures were not definitively identified.

5.3 Placental and Milk Transfer of DDAC

No data on placental or milk transfer were available for review.

5.4 Pharmacokinetics of DDAI

A study on the dermal and gastrointestinal absorption of ^{3}H-labelled didecyldimethylammonium iodide (DDAI) in rats was also located (84). DDAI is similar to DDAC, but the chloride ion is replaced by an iodide ion. Chemical behaviours of the two compounds are expected to be similar (84). The results indicate that the test compound does not readily penetrate cell membranes and that it is very stable and does not undergo degradation on the skin or in the stomach. Analysis of urine metabolites revealed that DDAI metabolism probably proceeds by oxidation and conjugation reactions analogous to the degradation of the fatty acids by B-oxidation. No $^{14}CO_2$ was detected in expired air, thus complete oxidation and N-dealkylation of the decyl group is not expected to occur.

6.0 TOXICOLOGICAL EFFECTS

6.1 Human Toxicity

Although the mammalian toxicity of quaternary ammonium compounds is not well established, several human fatalities have been ascribed to them (36). One source (1) listed a human oral LD50 of 450 mg DDAC/kg, and an acute dermal LD50 of 4300 mg/kg. These approximate human oral and dermal LD50 estimates are believed to be based on an extrapolation from undisclosed animal studies, although this was not stated in the source. A review of 10 human fatalities involving QACs concluded that the nature of the human toxic response varies widely with the dose and concentration of the substance, as well as with the route of administration and survival time of the victim (37).

6.2 Mammalian Toxicity

The following toxicity data are for various formulations of DDAC, as listed in Appendix B. All reported data have been converted to **100% active ingredient (AI)**, i.e. 100% DDAC, although it must be emphasized that the listed formulations were tested. Toxicity ratings, based on 100% active ingredients,

follow each oral and dermal toxicity value and reference, according to the table in Appendix C.

a) Acute Oral LD50:

Formulation	Species	Reported LD50	Converted (100% AI)	Toxicity
Bardac 2250	Rat	450 mg/kg (73)	225 mg/kg	4
Bardac 2280	Rat	450 mg/kg (2)	360 mg/kg	4
Timbercote II	Rat	1800 mg/kg (69)	360 mg/kg	4
F-2 Concentrate	Rat (males)	3500 mg/kg (68)	399 mg/kg	4
F-2 Concentrate	Rat (females)	2850 mg/kg (68)	325 mg/kg	4
100% DDAC	Rat	84 mg/kg (25)	84 mg/kg	4
100 % DDAC	Mouse	268 mg/kg (25)	268 mg/kg	4
5-25% DDAC	Rat	1190 mg/kg (38)	59.5-297.5 mg/kg	4

b) Acute Dermal LD50:

Formulation	Species	Reported LD50	Converted (100% AI)	Toxicity
Bardac 2280	Rabbit	>2000 mg/kg (2)	>1600 mg/kg	3
Bardac 2250/80	Rabbit	4350 mg/kg (80)	2175-3480 mg/kg	3
Bardac 2250/80	Rabbit (abraded skin)	3500 mg/kg (80)	1750-2800 mg/kg	3
80% DDAC	Rabbit	3342 mg/kg (64)	2674 mg/kg	3
Timbercote II	Rabbit	13,368 mg/kg (69)	2674 mg/kg	3
F-2 Concentrate	Rat	>2000 mg/kg (68)	>228 mg/kg	4

A subchronic dermal toxicity study in rats (16) involved application of 0, 0.1, 0.3 and 0.6% (w/w) DDAC solutions to the clipped backs of animals for 5 days per week over 13 weeks. The sites of administration were occluded for at least 6 hours on each day of dosing, after which time the dressings were removed and the application site rinsed with tap water and blotted dry. All solutions were administered at a constant volume of 2.0 ml/kg/day. The three concentrations therefore corresponded to dosage levels of 2, 6, and 12 mg/kg/day.

No treatment-related changes in clinical signs, food consumption, body weights, weight gain, ophthalmic parameters, hematology, clinical chemistry, gross pathology, or histopathology were observed. Mild skin irritation at the treatment site was observed in most animals at the 12 mg/kg/day level, in a few animals at the 6 mg/kg/day level, and in one female at the 2 mg/kg level.

c) Intraperitoneal LD50:

100% DDAC	Rat	45 mg/kg (25)
100% DDAC	Mouse	11 mg/kg (25)
100% DDAC	Guinea Pig	7 mg/kg (25) (LDLo*)

*Lowest published lethal dose

d) Intravenous LD50:

0.5 - 1.0% solution	Mouse	27 mg/kg (38)

f) Primary Skin Irritation: Rabbit — Severely irritating (2)

In an older study (67), a single application of technical grade (50%) DDAC to intact and abraded rabbits' skin caused severe erythema and edema after 24 and 72 hours on intact skin, and on skin surrounding the abraded area. Additional studies with the same product ("Aliquat 203") found that a 10% solution (5% DDAC) gave erythema, edema, and slight necrosis on rabbit skin (67). The non-irritating concentration was 0.01% (0.005% DDAC).

g) Sensitization:	Humans	Nonsensitizer (38)
	Guinea pigs	Nonsensitizer (68, 81)

h) Acute Inhalation LC50:

No studies were available for evaluation.

i) Feeding Studies:

A subchronic range finding study on mice involved dosing 15 animals/sex/group with 0, 100, 300, 600, 1000, or 3000 ppm DDAC for 89 days (males) or 90 days (females) (26). For the lowest four dose groups, these doses correspond respectively to mean intake levels of approximately 18, 52, 107, and 182 mg/kg for males, and 23, 68, 134, and 224 mg/kg for females. High mortality in the 3000 ppm groups prohibited calculation of daily intakes.

Treatment of mice with 3000 ppm DDAC in the diet for several days resulted in virtually 100% mortality in both sexes, with only one male surviving to termination of the study. Death was attributed to treatment-related severe wasting and dehydration resulting from gastrointestinal effects.

Treatment with 1000 ppm DDAC produced a 5% decrease in body weight in males with associated decreases in body weight gain. Similar depressed body weight in the females from this group was assumed to be related to DDAC exposure. No other changes were observed in males or females in the other dose groups. A clear definition of a maximally tolerated dose (MTD) for this experiment was not calculated due to the steep dose-response from virtually 100% mortality for mice in the 3000 ppm group to minimal body weight effects in the 1000 ppm group. The No Observed Effect Level (NOEL) for this experiment was 600 ppm (107-134 mg/kg/day).

In a similar 90 day subchronic oral toxicity study (15), Sprague-Dawley rats (15 rats/sex/dose) were exposed to DDAC at mean intake levels of 6, 18, 37, and 61 mg/kg/day for males, and 8, 22, 44, and 74 mg/kg/day for females (i.e. 100, 300, 600, and 1000 ppm groups, respectively). High mortality in the 3000 ppm group prohibited calculation of mean daily intakes.

Dietary exposure to 3000 ppm DDAC resulted in 80% mortality in both sexes. The three rats of each sex of this group that survived to the end of the experiment exhibited markedly reduced body weights, fluid- or gas-filled intestines at necropsy, and inflammation of the beginning of the large intestine (typhlitis). Pathological changes in clinical chemistry included decreased serum glucose and protein concentrations in both sexes, decreased albumin and globulin concentrations in females, and increased erythrocyte count and hemoglobin and hematocrit concentrations in males. These findings were considered to be related to the debilitated condition of the animals and/or the gastrointestinal lesions resulting from treatment. Experimentally induced fatalities were thought to result from gastrointestinal blockage and shock.

Administration of 1000 ppm or less of DDAC resulted in no treatment-related effects. The NOEL for DDAC in this strain of rat was therefore concluded to be 61-74 mg/kg/day.

In a recently completed chronic toxicity study (96), beagle dogs were dosed orally with 0, 3, 10, or 30 mg DDAC/kg/day (4/sex/group) for 52 weeks. During the first four and a half weeks of the study, several dogs in the 30 mg/kg/day dose group showed potentially life threatening decreases in body weight and food consumption. The dose was therefore decreased to 20 mg/kg/day. In some cases, depressions in weight and food consumption were so severe that the dogs in this group were removed from treatment completely during study days 31-36, and then reinstated at the 20 mg/kg/day dose level.

Results show that chronic administration of 3 to 20 mg DDAC/kg/day was not associated with mortality, changes in organ weights, gross pathological findings, ophthalmoscopic changes, or microscopic changes in selected organs and tissues. The 20 mg/kg/day dose was associated with decreases in mean erythrocyte counts; hemoglobin and hematocrit values; and mean total cholesterol, total protein, and albumin values. The 20 and 10 mg/kg/day doses were associated with an increased incidence of emesis, salivation, and soft/mucoid/liquid feces as compared to controls (96). The NOEL for systemic toxicity was considered to be 10 mg/kg/day.

In a chronic dietary toxicity/oncogenicity study (97), Sprague-Dawley rats received DDAC in the diet at concentrations of 0, 300, 750, or 1500 ppm for at least 104 weeks. These doses corresponded to approximate mean DDAC intakes of 13, 32, and 64 mg/kg/day for males, and 16, 41, and 83 mg/kg/day for females.

Treatment-related decreases in body weight and food consumption in both males and females were observed in the 1500 ppm group. In addition, possible treatment-related microscopic changes including hyperplasia of bile ducts in female rats and changes in mesenteric lymph nodes in male and female rats

related to blood in the sinuses were observed in the 1500 ppm treatment group. No treatment-related effects were seen in the type or incidence of clinical signs, survival, palpable masses, clinical pathology, organ weights, gross anatomic pathology, or ophthalmology. The NOEL for toxicity was considered to be 750 ppm. DDAC was not considered to be oncogenic under the conditions of this study.

A similar dietary oncogenicity study using CD-1 mice involved dosing animals with 0, 100, 500, or 1000 ppm DDAC for at least 78 weeks (98). Approximate mean intake levels were 15, 76.3, and 155.5 mg/kg/day for males, and 18.6, 93.1, and 193.1 mg/kg/day for females.

Treatment-related findings included decreased body weights and body weight gains in both males and females from the 1000 ppm groups. There were no treatment-related clinical signs of toxicity, increases in palpable masses, changes in food consumption, differences in organ weights or observations at necropsy, or differences in histopathological findings. The NOEL for this study was considered to be 500 ppm. DDAC was not considered to be carcinogenic under the conditions of this study.

For sake of completeness, a study is summarized here which the federal government of Canada (2) has indicated was seriously flawed. The government has stated the study is of little value in assessing the toxicological consequences of short term exposure, but reasons for dissatisfaction were not stated.

Dogs given doses of 5, 15, or 50 mg/kg (4 dogs/sex/group) of Bardac 22 (50% DDAC, 20% isopropanol, 30% water) for 13 weeks exhibited no toxic effects in any dose group (3). Behaviour, hematological, biochemical, urological, and pathological findings were within normal ranges throughout the study. The only treatment-correlated response found for any parameter was a slight weight depression in dogs given 50 mg/kg. A NOEL was not stated, but no toxic or pharmacologic effects were observed in any dose group.

6.3 Aquatic Toxicity

The following aquatic toxicity data are for various formulations of DDAC, as listed in Appendix B. All reported data have been converted to **100% active ingredient (AI)**, i.e. 100% DDAC, although less potent formulations may have been tested. Types of tests are indicated by: (f) - flow through bioassay, (s) - static bioassay, or (u) - unspecified bioassay. Toxicity ratings follow each 100% AI **96 hour** toxicity value and reference, according to the table in Appendix D.

24 hr LC50:

Concentration	Species	Reported 24 hr LC50	Converted(100% AI)
100% DDAC	Bluegill Sunfish	(u) 0.60 ppm (46)	0.60 ppm

Bardac 22	Rainbow Trout	(u) 1.25 ppm (70,75)	0.63 ppm

48 hr LC50:

Concentration	Species	Reported 24 hr LC50	Converted(100% AI)
Bardac 22	Catfish	(s) 4.8 ppm (71)	2.4 ppm
Bardac 22	Bluegill sunfish	(s) 0.75 ppm (71)	0.38 ppm
100% DDAC	Bluegill sunfish	(u) 0.30 ppm (46)	0.30 ppm
Bardac 22	Rainbow Trout	(u) 1.18 ppm (75	0.59 ppm
Bardac 22	Guppies	(u) 1.9 ppm (47)	0.95 ppm
100% DDAC	*Daphnia magna*	(s) 0.094 ppm (10)	0.094 ppm

96hr LC50:

Concentration	Species	Reported 24 hr LC50	Converted(100% AI)	Toxicity
Bardac 22	Catfish	(s) 2.6 ppm (71)	1.3 ppm	3
100% DDAC	Bluegill sunfish	(s) 0.32 ppm (11)	0.32 ppm	4
Bardac 22	Bluegill sunfish	(s) 0.59 ppm (71)	0.295 ppm	4
Bardac 22	Bluegill sunfish	(u) 0.54 ppm (44)	0.27 ppm	4
Bardac 2280	Rainbow Trout	(s) 0.880 ppm (41, 74)	0.704 ppm	4
Bardac 22	Rainbow Trout	(u) 1.1 ppm (45)	0.55 ppm	4
Timbercote II	Rainbow Trout	(s) 12.4 ppm (41)	2.48 ppm	3
F-2	Rainbow Trout	(s) 3.9 ppm (41)	0.44 ppm	4
F-2	Rainbow Trout	(s) 4.6 ppm (42)	0.52 ppm	4
assumed 100%	Rainbow Trout	(u) 1.24 ppm (2)	1.24 ppm	3
assumed 100%	Rainbow Trout	(u) 2.81 ppm (41)	2.81 ppm	3
100% DDAC	Coho Salmon	(s) 1.0 ppm (9)	1.0 ppm	3
F-2	Coho Salmon	(s) 5.9 ppm (43)	0.67 ppm	4
F-2	Chinook Salmon	(s) 3.2 ppm (64)	0.36 ppm	4
Bardac 22	Guppies	(u) 1.2 ppm (44, 47)	0.6 ppm	4
100% DDAC	Mysid Shrimp	(s) 0.069 ppm (8)	0.069 ppm	5

NOEC*	Species	100% DDAC
	Bluegill Sunfish	(s) 0.10 ppm (11)
	Coho Salmon	(s) 0.59 ppm (9)
	Mysid Shrimp	(s) 0.052 ppm (8)
	Daphnia magna	(s) 0.074 ppm (10)

* No Observed Effect Concentration

6.3.1 Bioconcentration

A bioconcentration study on Bluegill sunfish (*Lepomis macrochirus*) involved continuous exposure of fish to 59 ug of ^{14}C-DDAC/L of water for 28 days (27). This treatment was followed by an 18 day depuration period in which 40 fish were transferred to flowing, uncontaminated water. Radiometric analyses of water and selected fish tissues revealed that the concentration of ^{14}C-residues in nonedible, edible, and whole body tissues reached a steady state by day ten. Mean steady state bioconcentration factors for these tissues were 140x, 38x, and 81x, respectively.

Elimination of ^{14}C-residues from the selected tissue portions and consequently from whole body tissue was measured during the depuration period. Half-life of residues present on the final day of DDAC exposure was determined to be between 7 and 14 days of depuration. By day 14 of the depuration period, ^{14}C-residue concentrations present on the last day of exposure in the nonedible, edible, and whole fish tissues had been reduced by 71%, 57%, and 67%, respectively, based on tissue analysis of five fish. At the end of the depuration period (18 days), percentages eliminated for nonedible, edible, and whole body tissue were 66%, 38%, and 56%, respectively, compared to concentrations present at the beginning of depuration. Percent elimination appeared somewhat lower than at day 14 due to relatively high ^{14}C-residues in one of the fish sampled.

Skin tissue analysis after 28 days exposure showed ^{14}C levels approximately 2 to 6 times higher than those in edible tissues, indicating that there is significant binding of DDAC to the skin and scales of exposed bluegill sunfish. Based upon the short amount of time it took DDAC to reach steady-state and to depurate from tissues, along with the relatively low bioconcentration factor in edible tissues, DDAC would not be expected to bioconcentrate in fish tissues.

6.4 Avian Toxicity

<u>Acute Oral LD50:</u>		
Bardac 22?	Mallard Ducks	3300 mg/kg (70)
<u>8 Day Oral LC50:</u>		
Bardac 22	Bobwhite Quail	1950 ppm* (77)
100% DDAC	Bobwhite Quail	975 ppm (from 77)
Bardac 22	Mallard Duck	>3500 ppm* (77)
100% DDAC	Mallard Duck	>1750 ppm (from 77)

*mg/kg unspecified

6.5 Phytotoxicity

The toxic effect of QACs on the unicellular green alga *Chlorella*, and on a duckweed *Spirodela oligorhiza* has been investigated (48). In general, all types of QACs were strongly phytotoxic, with suppressed plant growth evident at concentrations above 10^{-5} M (approximately 3 to 5 ppm). Bardac 22 at 3 to 5 ppm suppressed plant growth in both species after 3 days. In duckweed, sublethal levels of QACs caused a yellowing or browning of the frond margins and the production of smaller sized fronds. In *Chlorella*, the size, shape, and internal organization of the cell were affected; death appeared to be due to disruption of chloroplast structure.

6.6 Comparative Toxicity

A comparison of some toxicity values for various antisapstain chemicals is shown in Table 1. From this table it is evident that, of the non-chlorophenol antisapstain chemicals, DDAC is the most toxic orally. However, it is one of the least aquatically toxic compounds. Consideration of the aquatic toxicity of DDAC is important since much of the stormwater effluent from lumber mills is discharged into rivers, lakes, or oceans, thereby exposing aquatic species to the chemicals used. Chemicals from leaching, spills, and other discharges also find their way into groundwater, and other bodies of water.

7.0 CARCINOGENICITY

Two carcinogenicity studies have been submitted by the manufacturer. In the first study (97), Sprague-Dawley rats received DDAC in the diet at concentrations of 0, 300, 750, or 1500 ppm for at least 104 weeks. These doses corresponded to approximate mean DDAC intakes of 13, 32, and 64 mg/kg/day for males, and 16, 41, and 83 mg/kg/day for females. DDAC was not considered to be oncogenic under the conditions of this study.

In the second study, CD-1 mice received 0, 100, 500, or 1000 ppm of DDAC in the diet for at least 78 weeks (98). Approximate mean intake levels were 15, 76.3, and 155.5 mg/kg/day for males, and 18.6, 93.1, and 193.1 mg/kg/day for females. Again, DDAC was not considered to be carcinogenic under the conditions of this study.

Presently, DDAC is not listed as a carcinogen by the National Toxicology Program, the International Agency for Research on Cancer, the Occupational Safety and Health Administration (40), or the Registry of Toxic Effects of Chemical Substances (RTECS).

8.0 MUTAGENICITY

Several studies evaluating the mutagenicity of DDAC have all given negative results. In the *in vitro* rat primary hepatocyte Unscheduled DNA Synthesis (UDS) assay (18), DDAC did not induce significant increases in UDS. Freshly prepared hepatocytes were exposed to concentrations of the chemical ranging from 0.05 ug/ml to 10 ug/ml. Concentrations ranging from 0.05 ug/ml to 2.00 ug/ml were selected for analysis of nuclear labelling and represented a good range of toxicity in both trials. None of the criteria used to indicate a UDS response was approached by the test material and no dose-related response was observed. The test material was therefore evaluated as inactive in this assay.

In a second mutation assay (19), the ability of 80% DDAC to induce forward mutations at the hypoxanthine-guanine phosphoribosyl transferase (HGPRT) locus in Chinese hamster ovary (CHO) cells was evaluated. Testing was performed with and without S9 metabolic activation. Mutant frequencies of all cultures treated with DDAC varied randomly with dose within the range acceptable for vehicle control mutant frequencies. Several random cultures did achieve statistical significance but were apparently due to normal assay variation and to vehicle control mutant frequencies that were on the lower end of the normal range. DDAC was therefore considered negative for mutagenic activity under the conditions of this assay.

Another study investigated the ability of DDAC to induce chromosomal aberrations in CHO's *in vitro* (72). Doses of 2, 4, 8, and 16 ug Bardac 22/ml were tested with activation, while doses of 1, 2, 4, and 8 ug/ml were tested without activation. Results indicated that Bardac 22 is not clastogenic in CHO cells *in vitro*, with or without metabolic activation.

In a *Salmonella typhimurium*/Ames assay, no mutagenic activity was observed when Bardac 22 was tested in strains TA 1535, TA 1537, TA 1538, TA 98, and TA 100 (82). Results were negative with and without metabolic activation. A bone marrow cytogenetic assay in rats treated with 600 mg/kg also gave negative results (1, 2).

One *in vivo* cytogenetic test assessed the effect of Bardac 22 on the incidence of chromosomal damage in rats (83). Rats were treated with the test compound by gastric intubation at a dose of 600 mg/kg bodyweight, then sacrificed at 6, 24, or 48 hours post dose. Results showed that Bardac 22 did not cause any statistically significant increases in the proportion of cells showing chromosomal damage at any of the three time points.

9.0 TERATOGENICITY AND REPRODUCTIVE EFFECTS

Two developmental toxicity studies, results of one teratogenicity study, and a two-generation reproduction study were located. In the first developmental toxicity study (28), sixteen mated New Zealand female white rabbits per dosage group were administered 0.0, 1.0, 3.0, or 10.0 mg/kg/day of DDAC by gavage at gestational days (gd) 6 through 18. The vehicle was deionized water.

Four of 16 does in the highest dose group died prior to gd 13. One doe at 10 mg/kg and two does at 1.0 mg/kg delivered early and were removed from the study. No females aborted. Maternal toxicity, including mortality, was evident at 10 mg/kg. Nonlethal indications of maternal toxicity were evident at 3 and 10 mg/kg, as evidenced by reduced weight gain and clinical signs during the treatment period. Developmental toxicity, including increased incidence of fetal

mortality and reduced fetal body weight per litter, were observed only at the highest dose. No teratogenicity was observed at any dose employed. The NOEL for maternal toxicity was set at 1.0 mg/kg/day, and the NOEL for developmental toxicity was set at 3.0 mg/kg/day.

The second developmental toxicity study involved dosing timed-pregnant Sprague-Dawley rats with DDAC on gestational days 6 through 15 (31). Twenty-five females per group received 1, 10, or 20 mg/kg/day by gavage.

No females died, aborted, delivered early, or were removed early from the study. Maternal toxicity was indicated at 10 and 20 mg/kg/day by characteristic clinical signs of audible respiration. Reductions in body weight and food consumption were also observed at 20 mg/kg/day during the treatment period. No evidence of developmental toxicity including teratogenicity was observed at any dose employed. The NOEL for maternal toxicity was 1.0 mg/kg/day. The NOEL for developmental toxicity was at least 20 mg/kg/day.

In female rats receiving 10, 25, or 50 mg/kg of Bardac 20, Bardac 22, or Bardac LF (percent DDAC unspecified) by oral gavage on days 6 to 15 of gestation, no teratogenic effects were observed (76). However, all formulations did cause several females to resorb one or more fetuses at the 50 mg/kg level.

A reproduction study (32) using Sprague-Dawley rats involved dietary administration of DDAC at target dosage levels of 0, 300, 750, or 1500 ppm for two generations. Dose levels in terms of mg/kg/day decreased throughout the study as animal body weight increased, and varied within and between dose groups and generations, so are not reported here.

A total of 28 males and 28 females were evaluated at each dose level. Animals were exposed to DDAC for 10 weeks prior to mating, and each of the two generations produced two litters. The original rats, called the F0 generation, were randomly paired within dose groups and mated over a three-week period to produce the F1A generation. Exposures continued through mating, gestation, parturition, and lactation. At least 10 days after weaning the F1A litters, F0 parents were mated in different male-female pairings, within dose groups, to produce the F1B generation. Exposures to DDAC again occurred from mating to lactation. After the F1B animals were weaned, F0 parents were necropsied and high dose and control animals were examined for histopathologic lesions.

Selected F1 parents were exposed to the same concentrations of DDAC as their parents for at least 10 weeks, and were then paired as described above to produce F2A and F2B generations. Mating, gestation, lactation, and necropsy of the F1 parents and selected F2A and F2B pups were performed as outlined above, except that no F2 animals were selected as parents.

Results indicate that continuous exposure to DDAC in the diet for two generations resulted in no adverse reproductive effects. Parental toxicity was observed at 1500 ppm (~112.6 mg/kg/day), limited to body weight reduction, weight gain depression, and decreased food consumption. Postnatal toxicity at 1500 ppm was indicated by reduced pup body weights. The NOEL for both adults and offspring was 750 ppm, indicating no increased risk to offspring in the absence of indications of adult toxicity.

10.0 IMPACTS OF OCCUPATIONAL EXPOSURE

Although DDAC has been used extensively in various applications, no quantitative occupational exposure data were located for any DDAC formulation. However, a review of F-2 concentrate (66) noted that sawmill workers in the mixing room could contact F-2 by splashing on the skin and into the eyes. It was also stated that most workers are potentially exposed to diluted treating solutions, mainly by skin contact while handling treated lumber. Workers near a poorly operating spray box or doing maintenance work on the box could be exposed to spray mist.

Information on human exposure to QACs has been fairly well documented (21). Since all QACs probably produce similar toxic effects (21), these are summarized here. Concentrated aqueous solutions (10% and sometimes less) are primary skin irritants, and concentrations as low as 0.1 to 0.5% are often irritating to conjunctivae and mucous membranes. Percutaneous absorption is probably insignificant.

Information on inhalation toxicity of the chemical was not found. Oral ingestion of strong aqueous solutions (10-20%) commonly produces superficial necrosis of mucous membranes with which they come in contact. Severe corrosion of the upper alimentary tract, and erosion, ulceration, and hemorrhage at the surface of the small intestine have also been observed.

Based on the concentrations involved in the above cases, the use of DDAC in the workplace is potentially hazardous. Inhalation was expected to be the main route of occupational exposure in one study (39), but air monitoring tests in several mills that use DDAC have shown a minimal risk. Because dermal exposure may also be a problem in mills, the use of proper protective clothing, including gloves, boots, and chemical goggles, and following proper chemical procedures are indicated.

An Acceptable Daily Intake (ADI) has been calculated by BC Environment. This value, derived in Appendix E, is 22.4 mg/day for a 70 kg person.

11.0 FOOD USE

DDAC is currently registered for a variety of antimicrobial uses under conditions where contamination of food products is unlikely (2). There are no data available in Health and Welfare Canada files concerning the potential food contamination resulting from the use of treated wood in the construction of food holding containers or storage facilities (2).

12.0 ANALYTICAL METHODS OF DETECTION

Two standard analytical methods for DDAC were supplied by Lonza (33). The first method involves detection of tertiary amines and alkyl chlorides by gas chromatography. The second method involves quantification of these compounds based on their solubility relationships in aqueous and chloroform layers. Detection limits were not stated but it appears that the method is used mainly for quantifying DDAC concentrations in NP-1 working dilutions, and for leachate and F-2 analyses (91).

Another method from Lonza (63) involves preparation of a disulphine blue indicator and its reaction with DDAC to form the disulphine blue-DDAC complex in chloroform. Chloroform solutions are then measured for DDAC levels by UV spectrometry using the absorption maximum at 625 nm for the disulphine blue-DDAC complex. Detection limits were not indicated.

An older method of analysis (35) used to measure DDAC in the range of 2-10% total chlorine involves combustion of the sample in a Parr oxygen bomb, with conversion of all chlorine present to an ionic form. The solution is then titrated to determine total chlorine in the sample.

Photolytic and hydrolytic studies sponsored by the manufacturer (12, 13) used thin layer chromatography (TLC) and liquid scintillation counting to measure ^{14}C-DDAC concentrations in water. Samples were spotted onto TLC plates, and the plates were then eluted in chloroform/methanol/formic acid to a distance of at least 10 cm above the origin. After elution, the plates were analyzed on a radio-TLC plate scanner, to locate the ^{14}C-active chromatographic zones. The detection limit was not given, but was at least 10 mg/L.

A QAC biodegradability study used a colourimetric test and UV spectrophotometry (34). For the colourimetric test, 1 ml of reagent (0.02% bromophenyl blue dye in alkaline water) was added to a 100 ml sample produced by adding 7.5% hydrochloric acid to shake flask samples. The reactant was then extracted with chloroform, read at 450 nm and 460 nm on a colourimeter, and measured for QAC concentration by comparison with standard curves prepared for each compound. Limit of detection for a 100 ml sample was 0.25 mg/L, well

below detection limits required for enforcing legislation in British Columbia (29). UV spectrometry involved subjecting refluxed samples to UV analysis at 263 and 268 nm and comparing results to specific standard curves to estimate concentrations.

Finally, a recent method developed by BC Research (87) involves treating field samples with hydrochloric acid, then adding a quaternary ammonium surrogate and extracting with dichloromethane. The raw extract is concentrated and a quaternary ammonium performance standard is added to the final extract volume. The extract is then examined on a gas chromatograph equipped with a nitrogen/phosphorus specific detector (NPD). The detection limit is 0.025 mg/L.

13.0 EXISTING LEGISLATION

In British Columbia, provincial regulations (29) state that, from September 1, 1990 forward, the concentration of DDAC in effluent shall not exceed 700 ug/L. A stormwater effluent standard is derived in Appendix F.

Provincial regulations also stipulate that the rate of emissions to the air from chemical spray booth stacks is not legally permitted to exceed 7.0 mg DDAC/second. A derivation of an interim emission standard is shown in Appendix G. No other standards were located.

14.0 CONCLUSION

The use of DDAC in the lumber industry has come about relatively recently, but the compound has a long history of use in other areas. Thus, the toxicological and environmental impact database for DDAC is extensive. Several long-term toxicity, carcinogenicity, reproduction, and developmental toxicity studies have recently been completed, as have environmental degradation studies. These studies answer some questions in terms of its use as an antisapstain chemical.

In laboratory studies, DDAC has been shown to be hydrolytically and photolytically stable. In studies involving wood treatment, DDAC levels as high as 76 ppm have been found in the initial leachates. The concentration of DDAC in later leachates are in the 6 ppm range. These data indicate that there is a potential for significant quantities to enter the environment. Soil adsorption/desorption studies have shown that DDAC is relatively immobile in soil, suggesting that there is little likelihood of ground water contamination from surface soil contamination. The data regarding microbial degradation of DDAC indicate that both the concentration of DDAC, and the type or quantity of microbial organisms initially present are important factors in determining the ultimate fate of DDAC in soil or sediment.

DDAC is moderately toxic to very toxic to mammals, depending on route of administration. It appears to be excreted rapidly, with only low levels remaining in tissues after one week. In terms of aquatic exposure, DDAC is generally moderately to very toxic to fish species. It has been shown to be highly toxic to an alga and to a duckweed, but implications of this toxicity are unknown.

For workers exposed to DDAC or its formulations, the main hazard appears to be its potential to cause severe skin and eye irritation. Importantly, DDAC has not been shown to be mutagenic, carcinogenic, or teratogenic in several laboratory studies. However, further exposure studies should be done to assess the potential occupational impacts of the compound and its formulations.

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Table 1: A comparison of toxicity values of antisapstain chemicals

	Oral LD50* Rat - mg/kg	Dermal LD50* Rabbit - mg/kg	Aquatic 96hr LC50* Fish species**-ppm
DDAC	84[25]	>1,600[2]	0.44[41] Bluegills 0.27[44]
IPBC	1,426[50]	>1,940[50]	0.065[50]
TCMTB	360[55]	324[55]	0.017[55] Chinook 0.015[55]
Copper-8	702[5]	>1,960[5]	0.00016[5]
Sodium carbonate	4,000[56]	not avail.	100[56]
Sodium borate	2,660[56]	>2000[56]	377[56]
NaTCP	210[57]	270[57]	0.3-0.13[57]
NaPCP	71[50]	250[51]	0.048[52] Coho Salmon 0.032[54]
PCP	27[51]	39[50]	0.044[53] Bluegills 0.020[53]

*Values quoted are the lowest measured for each species, and are based on 100% active ingredients, although less potent formulations may have been tested.

**Fish species is Rainbow trout unless otherwise noted.

APPENDIX A

DATABASES SEARCHED FOR INFORMATION ON DDAC

The following computerized databases were searched for information on DDAC:

CA Search
Toxline
Toxlit
AQUIRE
Medline
RTECS
CCOHS

APPENDIX B

PRODUCTS CONTAINING DDAC, AND THEIR MANUFACTURERS

PRODUCT	%DDAC	MANUFACTURER
Bardac 22/2250	50%	Lonza, Inc., Fair Lawn, NJ
Bardac 2280	80%	Lonza, Inc., Fair Lawn, NJ
NP-1	64.8%	Kop-Coat Inc., Pittsburgh, PA
Timbercote II	20%	Napier Pacific Industries Inc. Surrey, BC
F-2	11.4%	Walker Brothers, Burnaby, BC
Ecobrite III	2%	Canfor, Vancouver, BC

APPENDIX C

MAMMALIAN LETHAL TOXICITY CLASSES FOR CHEMICAL COMPOUNDS*

Toxicity Rating	Commonly Used Term	Oral/Dermal** (mg/kg)	Inhalation‡ (ppm-mg/L)
1	Practically Nontoxic	>15,000	>200
2	Slightly Toxic	5,000-15,000	20-200
3	Moderately Toxic	500-5,000	2-20
4	Very Toxic	50-500	0.2-2
5	Extremely Toxic	5-50	0.02-0.2
6	Ultra Toxic	<5	<0.02

*Toxicity ratings adapted from references 21, 85, and 4.

**Index: acute oral or dermal LD50.

‡Index: LC50 - four hour inhalation exposure.

APPENDIX D

AQUATIC LETHAL TOXICITY CLASSES FOR CHEMICAL COMPOUNDS*

Toxicity Rating	Commonly Used Term	Exposure Conc.** (ppm - mg/L)
1	Practically Nontoxic	>100
2	Slightly Toxic	10 to 100
3	Moderately Toxic	1 to 10
4	Very Toxic	0.1 to 1
5	Extremely Toxic	0.01 to 0.1
6	Ultra Toxic	<0.01

*Toxicity ratings adapted in consultation with reference 86.

**Index: 96 hour LC50 or EC50.

APPENDIX E

Calculation of an Acceptable Daily Intake (ADI) of DDAC for Humans

Following a method used by Fox (58), an ADI may be derived for humans based on the 32 mg/kg/day NOEL in male rats in the 104 week chronic dietary toxicity/oncogenicity study (97):

Calculation of human ADI based on rat NOEL

a) NOEL from chronic rat: 32 mg/kg/day (male rats)

a) Safety factor used: 100x

i.e., -100x for conversion of chronic animal to human data

b) Human DDAC ADI: = 32 mg/kg/day/100
= 0.32 mg/kg/day

c) For a 70 kg adult human: = 0.32 mg/kg/day x 70 kg
= 22.4 mg/day ADI

APPENDIX F

Derivation of Stormwater Effluent Criteria for DDAC

Adapting a method used by Fox (see Reference 17 for more details), the formula for calculation of a stormwater discharge criteria is as follows:

Criteria = 96 hour LC50 x AF x EF x DMF x DF

where:
- AF = acute toxicity safety factor (1.0)
- EF = environmental fate safety factor (1.0)
- DMF = degradation product and metabolite hazards safety factor (1.0)
- DF = dilution factor (1.0)

The 96 hour LC50 used is the value for the most sensitive salmonid species, i.e. Rainbow trout. Fox used the value of 880 ppb for 80% DDAC (41), to give a value of 704 ppb total active ingredients. Provincial antisapstain legislation limits effluent levels to 700 ppb (ug/L) (29).

APPENDIX G

Calculation of an Air Emission Guideline for DDAC

Based on the ADI given in Appendix E, an emission guideline is calculated below. A method used by Fox (58) is followed.

I. Assumptions

Adult
daily respiration = a) 20 m^3*
mean body weight = 70 kg
absorbs 100% of inhaled DDAC
respiration is only route of exposure

*This respiration value is based on values from the US Environmental Protection Agency (88).

II. Conversion of DDAC ADI to $ug/m^3/day$

Adult ADI_{70}

ADI = 22.4 mg/20 m^3/day
= 1.12 mg/m^3/day
= 1120 ug/m^3/day (24 hour ambient air guideline)

III. Conversion of 24 hour ambient air guideline to 1 hour ambient air guideline

For use in subsequent dispersion modelling calculations, the 24 hour ambient air guideline should be converted to a 1 hour ambient air guideline. Using the formula (59):

$$C2 = C1 \times (T1/T2)^a$$

where C2 = 1 hour average concentration
C1 = concentration at time T1
T1 = time period of the original standard
T2 = 1 hour
a = exponent = 0.28

then the one hour ambient air guideline is as follows:

$$C2 = 1120 \times (24/1)^{0.28}$$
$$= 2727 \text{ ug/m}^3\text{/hour}$$

IV. Calculation of an Emission Guideline through Dispersion Modelling

Given meteorology data and stack characteristics, at an emission rate of 1 g DDAC/sec, maximum ground level concentration of DDAC will be 0.00019 g/m^3. For a maximum ground level concentration of 1 g/m^3 to arise, an emission rate of ~5000 g/sec would have to occur. For a maximum ground concentration of 2727 ug/m^3 to arise, an emission rate of 13.64 g/sec would be required:

$$5000 \text{ g/sec} \times 2.727 \times 10^{-3} \text{ g/m}^3 = 13.64 \text{ g/sec.}$$

Using an engineering safety factor of 35, as used to develop an emission guideline for chlorophenates (60), an appropriate guideline for DDAC is as follows:

$$13.64/35 = 0.39 \text{ g/sec}$$
$$= 390 \text{ mg/sec}$$

N.B. Existing provincial legislation (29) allows spray booth emissions of 7.0 mg/second, based on capabilities of available technology.